Dizziness

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Introduction: The problem of dizziness is one of the most exasperating in the practice of medicine. Physicians all know that sinking feeling elicited by the patient who sits down and, when one asks, "What can I do for you?" says, "I'm dizzy." The goal of this paper is to offer medical practitioners a reasoned approach to dizziness that will lead expeditiously to diagnosis and effective therapy.

Principles of Diagnosis

The first principle in evaluating a dizzy patient is to take an open ended history. This is a good rule in taking any medical history, but it is particularly applicable in this instance. When the patient says to you, "I am dizzy," sit back in your chair, slowly spin around, perhaps stare aimlessly out the window, and reply, "What do you mean, dizzy?" Then wait for the response. This may take what seems to be a long time; nonetheless, don't ask, "Does the room spin?" "Do your legs get weak?" "Do you feel as if you might stagger?" "Are you lightheaded?" because the answer to all these questions will nearly always be yes. If you are fortunate enough to be the first physician to examine a patient complaining of dizziness, always take the undirected approach. Merely say to the patient, "What do you mean, dizzy?" and wait for the response. There are several possible responses.

Syncope or near-syncope

"I feel as if I might faint," or "I feel giddy or light-headed." Some patients do faint or report that they have done so while others have never actually fainted (near-syncope). Pathophysiologically, both syndromes suggest any of several cardiovascular disorders that produce a generalized decrease in cerebral blood flow. There is no qualitative difference between syncope and near-syncope with respect to the differential diagnosis.

Circulatory syndromes that should be considered include *orthostatic hypotension,* which may have a number of causes, many of them iatrogenic (e.g., antihypertensive agents and/or vasodilators). Volume depletion is also a common cause of this syndrome. Over-the-counter vasodilators, such as alcohol, should be considered and environmental phenomena, such as high ambient temperature and excitement leading to hyperventilation may all be part of the problem. A rise in the heart rate without a fall in blood pressure, the postural orthostatic tachycardia syndrome (POTS) has two major categories of causes: mild autonomic failure of any cause and anxiety on stanging. *Cardiac arrhythmias* are a very infrequent cause of syncope and near-syncope, however dizziness during or just following exercise should raise the specter of a cardiac problem, such an arrhythmia or left ventricular outflow obstruction (e.g. aortic stenosis or asymmetric septal hypertrophy). If the history suggests arrhythmic episodes, holter monitoring and even long-term loop monitoring of the cardiac rhythm may be required. Techniques are now available that allow continuous monitoring of the electrocardiogram for long periods of time (weeks) via a wireless transmitter, held with a bandaid-like device secured to the chest wall. An event button is available for the patient to mark episodes of dizziness. *Hypersensitive carotid sinus is* relatively uncommon. *Neurocardiogenic* syncope and near syncope*,* otherwise known as vasovagal syncope my be called the swoon or faint by patients. Neurocardiogenic syncope is probably due to over activity of the baroreceptor reflex such that brief periods of hypertension result in disproportionate bradycardia and hypotension resulting in decreased cerebral blood flow and consequent loss of consciousness. Any failure of

the autonomic reflex necessary to maintain cerebral blood flow in the upright posture can cause dizziness caused by near syncope. High ambient temperature, emotional excitement and natural vasodilators, such as alcohol, all may disable the systemic vasoconstriction, mediated by sympathetic alpha receptors, that allows for preservation of cerebral blood flow with orthostatic stress.

Disequilibrium

"I feel as if I might fall." This version of dizziness generally reflects one of two major categories of neurologic disease, apart from disorders of the vestibular system.

Cerebellar ataxia is due either to a primary disease of the cerebellum (e.g., cerebellar degeneration, tumor in or near the cerebellum, cerebellar infarct) or disorders of the tracts leading to (cerebellopetal) or from (cerebellofugal) the cerebellum . Neurologic examination will ordinarily reveal such pathology by revealing axial (e.g. wide based gait; falling to one side) or appendicular (e.g. side to side tremor on goal directed action).

The multiple sensory deficits syndrome is due to several abnormalities in the various sensory proprioceptive systems. When a number of these systems fail, the central nervous system receives conflicting proprioceptive input, with consequent dizziness. The typical patient is rather elderly, perhaps with some *visual disorder* due to cataracts, some *auditory disorder* due to presbyacusis, some myelopathy, perhaps related to cervical spondylosis or cobalamin deficiency, and *peripheral neuropathy* due to diabetes and/or chronic use of alcohol. Such a patient typically complains of dizziness at night, for instance, when the lights are out or dim and he or she has to go to the bathroom. On occasion, the patient may fall, particularly in environments in which there are no reliable visual cues (e.g. the shower).

The treatment of this extremely common syndrome is common sense. As many of the sensory abnormalities that can be corrected, should be. Cataracts and hearing disorders can be treated, and the progression of some peripheral neuropathies can be prevented by abstinence from alcohol. One might also advise the patient to keep the lights on at night, which would help the visual system compensate for other sensory abnormalities. Such patients should not be treated with drugs that might sedate them, as antivertigo medications would do. Mistaking this syndrome for vertigo would, in fact, make matters worse.

Anxiety and/or depression

There are patients who when asked, "What do you mean, dizzy?" respond, usually after a pause, "Dizzy." If the physician persists with "Do you mean you might faint?" or "Do you mean that you might fall?" or "Do you mean that the room spins?" the patient repeats, "No, I mean I'm dizzy." This disorder can only be called true dizziness, and it generally arises from various psychological disorders, most commonly anxiety (with or without hyperventilation) and/or depression.

Affective disorders can often be recognized because of the effect that the patient has on the examiner’s mood. If one feel depressed or anxious oneself after spending time with a patient, it may well be because the patient is depressed or anxious. It is extremely important to recognize instances when dizziness represents a metaphor for anxiety or depression, because treatment for vertigo is likely to exacerbate these disorders, whereas treatment for depression and anxiety might dramatically relieve the dizziness.

Vertigo

The fourth and last category of disorder found in patients who complain of dizziness is true vertigo (an illusion or hallucination of motion). Some patients insist that they themselves are moving, others that the environment is moving. In either case the patient transmits the message that they feel as if they are tilting, rocking, falling, spinning or moving in some other fashion.

Vertigo indicates a disturbance in the vestibular system, which is responsible for keeping the central nervous system informed of the head's position in space, its relation to the pull of gravity, and its acceleration in various planes. The important clinical question is whether the vertigo is due to a disorder in the *peripheral nervous system* (the end organ or the peripheral nerve) or in the *central nervous system* (the brainstem or its projections to parts of the cerebral cortex, particularly the temporal lobe). Each lesion has its own differential diagnosis and treatment.

Evaluation of Vertigo

The first step is to perform a complete history and physical examination and a neurological examination with particular attention to the VIII cranial nerve. The VIII cranial nerve is in fact two separate cranial nerves, the vestibular and cochlear. These two nerves have closely juxtaposed end organs, run closely together in the internal auditory meatus, and have two completely different pathways in the central nervous system. Because of the close proximity of these two nerves and their end organs, it is common for disease of one to affect the other. *The physician should therefore examine both aspects of the VIII cranial nerve whenever there is a complaint of vertigo.*

Cochlear VIII Nerve Function

Pure tone hearing loss

Auditory testing: Examination of the cochlear system involves three steps whether or not the patient complains of hearing loss. The first is to test for pure tone hearing loss. This can be done in the office by comparing the sensitivity of the patient's ears or comparing the patient's ears with one's own, using a ticking watch or the sound of fingers rubbing together.

Sensory neural vs conductive hearing loss

If there is a hearing loss, the next step is to determine whether it is a sensory neural hearing loss (i.e., a neurologic problem), or a conductive hearing loss, (i.e. a disorder in the middle ear interfering with the functions of the ossicles). These determinations are made by using two tests: the Weber and Rinne.

The Weber test is performed by placing a vibrating tuning fork at the midline of the skull and asking the patient on which side the sound can be heard. If there is a definite lateralization to one side, one can determine whether there is sensory neural or conductive hearing loss. For example, if the Weber lateralizes to the left, this may be interpreted as either a left-sided conductive hearing loss or right-sided sensory neural hearing loss. *Combining this information with the knowledge of which ear has the hearing loss,one can determine whether that loss is sensory neural on the right or conductive on the left.*

The Rinne test is easy to apply in an office setting. Bone and air conduction are compared by placing the tuning fork first over the mastoid bone and then in front of the ear, asking the patient which is louder. Under normal circumstances, air conduction is better, because the ossicles in the middle ear amplify and transmit the impulse through the middle ear to the inner ear. If the ossicles are not functioning because of otosclerosis, cholesteatoma, or fluid in the middle ear, air conduction may suffer, which leads to a situation in which air and bone conduction are equal or bone

conduction is the better of the two. If, however, there is sensory neural hearing loss, air conduction remains better than bone conduction.

Cochlear vs Retrocochlear Hearing Loss

The third step in the hearing examination, needed only if there is a sensory neural loss, is perhaps the most important of the differential procedures but paradoxically the one least well known to many physicians. The issue is whether the sensory neural deficit is due to end organ disease (cochlear) or to disease of the nerve or the central nervous system (retrocochlear).

Speech discrimination testing can be done in the office to differentiate a cochlear from retrocochlear sensory neural hearing loss. There are a number of ways to make this distinction, but many require the services of an audiologist. The physician whispers words in the affected ear (e.g., hot dog, ice cream) loud enough for the patient to hear. At the same time, a sound is made in the other ear so that the patient cannot hear the words using the unaffected ear. Putting a finger in the patient's other ear and moving it around will serve the purpose. This is done on both sides several times, having the patient repeat the words each time, and comparing the two ears. One can also use the telephone for this purpose, testing each ear separately for words delivered through a telephone handset.

In people with cochlear-type sensory neural hearing loss, such as occurs in Ménière disease, speech discrimination is not perfect, but it is *relatively* preserved. On the other hand, in patients with retrocochlear hearing loss, such as accompanies a vestibular Schwannoma, there is a disproportionate loss of speech discrimination. Thus a patient with a cochlear hearing loss should be able to understand 70% or more of the words heard, whereas a patient with a retrocochlear hearing loss might understand only two out of ten words. *If there is any question of a retrocochlear hearing loss, one may order an audiogram or go directly to an MRI to image the VIII cranial nerve..*

Vestibular VIII Nerve-Function

Testing for nystagmus: The vestibular aspect of the VIII cranial nerve may be examined by testing for nystagmus. First, ask the patient to sit on the end of the examining table and to look about 45° to the right and to the left. (Asking the patient to look beyond 45° is not useful, since when asked to look too far in either direction, about 10% of the normal population show some degree of gaze-evoked end-point nystagmus.) If nystagmus develops when the gaze is directed to 45°, note the direction of the fast phase, the direction of the slow phase, and in what position of the eyes they occur.

Next the patient should be put through a series of positions called the Dix-Hallpike maneuver, in which the head is hung over the end of the examination table with the head turned first in one direction and then in the other. One should determine whether the vertigo is elicited by the maneuver and, if so, whether there is an associated nystagmus. It is often useful to use a light magnifying glass as the nystagmus may be of short duration and small amplitude. All vertigo is positional to some extent, but if vertigo is positional only, there are specific pathogenetic and prognostic implications. Once position testing has been done, the physician knows in which direction the world seems to be spinning and in which direction the patient seems to be falling when the vertigo develops. The directions of the fast and slow phases of the nystagmus have been recorded. Perhipheral lesions produce nystagmus in one direction in a single position of the head, whereas central disorders may produce bi-directional nystagmus in a single position of the head. For example, if one sees the fast phase of nystagmus beating rightward on looking rightward and beating leftward on looking leftward, all in the same position of the head, a central nervous system cause is likely. Note the peripheral is not synonymous with good and central with bad. For example, the use of benzodiazepine drugs will produce bi-directional nystagmus in a single position of the head. The cause is central intoxication of the neural integrator in the eye movement control

centers in the pons, but it is benign and easily managed. On the other hand, a tumor arising from the vestibular nerve (e.g. vestibular Schwannoma) is a serious, but peripheral disorder.

Next the horizontal head thrust maneuver should be performed. Ask the patient to fix their gaze on your nose and rapidly turn the head about 30% first in one direction and then the other. The vestibular-ocular reflex (VOR) tends to turn the eyes in the opposite direction of the head, so as to maintain fixation on the object of regard. During consciousness, this reflex is inhibited by the rapid eye movement pathway arising from the frontal eye fields and descending to the opposite pontine paramedian reticular formation. In order to test the intactness of the vestibular ocular reflex in a conscious person, one must occupy the frontal-pontine pathway which is responsible for the VOR's inhibition. Fixation on an object serves this purpose. Thus, when the patient fixes on the examiner's eyes and the head is turned horizontally, the eyes remain on the object of regard (the examiner's nose). If the vestibular ocular reflex is disordered on one side (as in vestibular neuritis), when the head is thrust in the direction of the lesion, the eyes do not stay on the target, rather moving slightly in the direction of the head turn. One will then see a refixation saccade back to the object of regard (the examiner's nose). When the horizontal head thrust maneuver is abnormal in one direction, it is very likely that there is a lesion in the peripheral part of the vestibular system on one side. Absence of a refixation saccade in either direction in a vertiginous patient suggest a central cause.

Finally, one should test for a vertical skew deviation, a finding that is highly suggestive of a central disorder. This is done by alternately covering the eyes and looking for a vertical refixation saccade as the occuder is moved from one eye to the other.

Peripheral or Central Nervous System?

Some knowledge of the neuroanatomy and neurophysiology of the vestibular system is required to interpret the data obtained from the neurological examination.

Vestibulo-ocular reflex

The end organ of the vestibular nerve is located in the semicircular ducts, utricle, and saccule. The lateral, or horizontal, semicircular duct is oriented in the inner ear so that it tilts at about 30° above the horizontal plane. When the head is held in the usual carrying position, this duct is approximately parallel to the ground. Thus turning the head right and left would be expressed almost entirely in a vector within the plane of the lateral semicircular duct.

When the head turns to the left, a series of impulses is initiated (beginning with stimulation of the hair cells in the left lateral semicircular duct) that leads to contraction of the right lateral rectus muscle (right eye abductor). This sequence, taken no further, would of course lead to a situation in which the eyes are pointed in two different directions, which would produce diplopia. Thus, a corresponding series of impulses must also reach the left medial rectus muscle in order for the left eye adductor to contract as well.

In a comatose patient with an intact brainstem but with cortical signals in abeyance, the vestibulo-ocular reflex can be elicited by turning the patient's head, which produces the *oculocephalic reflex,* or the so-called doll's eyes. In a conscious patient the reflex may be demonstrated by having the patient fix his or her gaze on a distant object or by infusing the ears with warm or cold water (the *caloric reflex).* Although the caloric reflex should be a routine part of the evaluation of a comatose patient, in an awake patient it is a procedure perhaps best left to the otologist or oto-neurologist.

Cerebral cortex

The movement of images on the retina sends information to the occipital cortex through the usual visual pathways. In addition, it is presumed that information regarding the movement of the eyes may reach the cerebral cortex even in the absence of visual stimuli, as proprioceptive organs in the orbit probably convey information to the parietal cortex.

The cerebral cortex is faced with an apparent paradox. The frontal eye fields could, of course, hve turned the eyes to produce a voluntary saccade (rapid conjugate eye movement) to the right. It is also possible that the right parietal-occipital region could have turned the eyes to the right by producing a conjugate pursuit or tracking eye movement, but in this case. If however, these areas have not, in fact, fired the cerebral cortex has received conflicting information (i.e. proprioceptive information indicates that the eyes have turned, but there is no indication that either a saccade or pursuit command was generated.

What conclusion can the cerebral cortex draw? It concludes not that the eyes have moved to the right but that the world has moved to the left! This erroneous conclusion is based on conflicting information. The resultant sensation of vertigo is thus usually a misperception of a stimulus (illusion). Vertigo may also occur as the result of a perception without a stimulus (hallucination). Migrainous vertigo and epileptic vertigo would be examples of hallucinatory vertigo.

Frontal lobe

The frontal lobe makes a correction for the abnormal eye movement that was generated by the disabled vestibular dysfunction. The corrective phase of the vestibulo-ocular reflex arises from the frontal eye fields and results in rapid turning of the eyes back to the left.

Thus a stimulus arising from the left vestibular system causes a slow conjugate eye movement to the right followed by intermittent rapid conjugate correction back to the left. It is associated with a vertigo in which the patient has a feeling that the world is spinning to the left while he is being pulled to the right.

The patient's feeling of being pulled may become worse when the eyes are closed, because closing the eyes removes another proprioceptive system that would help to compensate. *Romberg's sign (* i.e., a patient's balance is seen to become worse with the eyes closed), may be seen *in any abnormality producing a proprioceptive disorder,* including peripheral neuropathy and disease of the spinal cord as well as disease of the vestibular system.

The two phases of nystagmus

Thus vestibular imbalance nystagmus consists of two components. The first (active) phase originates in the brainstem or vestibular system, is caused by different vestibular input from the ears, and is associated with slow eye movement. The second (corrective) phase is initiated by the frontal eye fields in the cerebral cortex and is associated with fast eye movements. Both phases act through the final common pathway of the ocular motor system of the brainstem.

Under normal circumstances, the entire vestibular system functions bilaterally with all of its central connections. There is no vertigo or nystagmus with ordinary accelerations of the head.

Criteria for locating the lesion

There are four criteria for a peripheral type of vertigo and nystagmus . If there are 1) fast-phase nystagmus away from the lesion, 2) slow-phase nystagmus toward the lesion, 3) environment spinning away from the lesion, and 4) Romberg's sign toward the lesion, one can say with

confidence that there is a lesion of the peripheral nervous system, probably in either the end organ or the peripheral nerve.

If any of these four rules fails to hold, one can assume by exclusion that the lesion is in the central nervous system. Central nervous system lesions can cause bilateral nystagmus in the same position of the head, vertical nystagmus of any kind, and any conditions in which the directions of the fast and slow phases, the Romberg's sign, and the spinning of the environment do not strictly fit the four criteria specified. Those criteria specify only the anatomic localization without implying anything about the severity or seriousness of the underlying disease. Peripheral diseases can be self-limiting (e.g., vestibular neuronitis) or very serious (e.g., vestibular schwannoma). Central diseases can range from the trivial complications of many drugs (e.g. benzodiazepines) to vertebrobasilar insufficiency with our without brainstem and/or cerebellar stroke.

Synthesizing the data

Thus by testing the auditory system and the vestibular svstem, one can divide all cases of vertigo into three categories: *1)peripheral* (by vestibular criteria) *cochlear disease* (by auditory criteria and signs), 2) *peripheral* (by vestibular criteria) *retrocochlear disease* (by auditory criteria), and 3) *central disease.* With this in mind, we can now consider the major diseases in each category.

Peripheral Cochlear Lesions

Labyrinthitis is thought to be a result of viral infection of the endolymph and perilymph affecting both the vestibular and cochlear components of the system. The usual history is a viral illness followed by acute onset of severe spinning vertigo and sensory neural deafness with tinnitus. Examination shows a typical peripheral picture by vestibular criteria and a classic cochlear picture by auditory criteria. The horizontal head thrust maneuver will be abnormal in one direction. The nystagmus will be unidirectional in a single position of the head and there will be no vertical nystagmus nor vertical skew, the latter tested by the alternate cover test. Despite its severe onset, labyrinthitis is a benign illness, which resolves completely in three to six weeks. Patients regain normal hearing and vestibular function.

Vestibular neuritis, or acute vestibulopathy, is thought to be pathogenetically identical to labyrinthitis but without any hearing symptomatology. If the patient has vertigo unaccompanied by a hearing abnormality, it is strictly speaking impossible to be sure whether the disease is cochlear or retrocochlear. However, its natural history is also benign, and it resolves completely in three to six weeks, which makes a retrocochlear illness very unlikely.

Cochlear neuritis is the syndrome of acute pure deafness without vestibular symptoms or signs. It is thought to be analogous to vestibular neuritis.

Ménière disease is caused by a cryptogenic hydrops of the endolymph such that there is intermittent swelling of the semicircular ducts, with damage to the hair cells. An attack of Ménière syndrome is typically characterized by a dull ache in the region of the mastoid process or around the ear associated with severe tinnitus, a cochlear kind of sensory neural hearing loss, and a typical peripheral type of vestibular syndrome with severe spinning vertigo. It is identical in almost every respect with an acute attack of labyrinthitis. However, it does not resolve completely in three to six weeks, and patients are left with residual hearing loss. Several months or years later a similar attack may occur, leaving the patient with even more severe hearing loss. Tinnitus, a nonspecific sign of auditory system disorder, is a major problem for these patients, who can be terribly disabled for weeks at a time by the vertigo that accompanies acute attacks.

Many therapies have been tried, including shunting of the perilymphatic system and diuretics, but none are curative. About 15% of these patients have bilateral disease in subsequent years.

Management of such patients is complex and often best entrusted to an otolaryngologist or otoneurologist, as deliberate toxic (e.g. intraaural aminoglycoside antibiotics infusion) destruction or surgical severing of the vestibular nerve may be required.

Benign positional vertigo, or Bárány's vertigo, usually occurs in older patients and is characterized by the sudden onset of a peripheral vestibular syndrome *with no auditory aspect.* It is present only in certain positions, which are specific to the individual.

Typically, the patient reports that a few moments after attaining a certain position, perhaps in bed at night, severe vertigo occurs in which the world spins in one direction while the patient has a sensation of falling in the other direction. If he or she does not move, the vertigo stops, which implies that it is transient in type. If the patient sits up, the vertigo recurs, but this time in reverse. If the patient repeats the posture several times, the tendency toward vertigo and nystagmus will fade. All the symptoms can be reproduced using the Dix-Hallpike maneuver, during which the patients will experience vertigo with the affected ear down and an associated nystagmus that is rotatory (tortional) in the dependent eye and vertical in the opposite eye. Benign positional vertigo has a benign natural history, which improves gradually over a six-month period and ends with complete recovery.

Etiology of Benign Positional Vertigo

Canalolithiasis is one of two causes to explain benign positional vertigo. The theory of canalolithiasis maintains that bits of calcium break off from the otolithic apparatus in the ear, perhaps as a consequence of aging or minor head trauma. If these bits of calcium are floating in the posterior vertical canal's endolymph, they will fall with gravity, which initiates an impulse arise from the dependent ear. Since the calcium tends to fall into the most dependent of the three semicircular ducts, the canalolithiasis tends to affect the posterior vertical semicircular duct resulting in the characteristic vertigo and nystagmus pattern only when the affected ear is down. Canalolithiasis of the horizontal canal is relatively rare and of the anterior vertical canal unknown, probably simply due to the proximity of posterior and, to a lesser extent, the horizontal canal to the utricle from whence the otolithic debris arise.

Perilymphatic fistula is a rarer cause of positional vertigo. Normally the middle ear and inner ear are separated by the oval and round windows, which are completely sealed. If for some reason (e.g., head trauma) a crack develops in the oval or round window, some of the perilymph may leak from the inner ear into the middle ear. Such patients may have intermittent episodes of conductive hearing loss superimposed on a sensory neural hearing loss.

Superior Canal Dehiscence is another fistula in which the leak comes from the superior vertical canal. This unusual syndrome causes the characteristic sign of vertigo and nystagmus being exacerbated by sound (Tullio phenomenon).

The presence of a fistula may be detected by placing the otoscope in the ear and closing the glass window, which produces an air-tight space. Air is then pumped into the external ear using the balloon attachment to the otoscope. This air distorts the tympanic membrane, which briefly increases the pressure in the middle ear. Under normal circumstances, a mild sensation in the ear is produced but no vertigo. If, however, there is a pathologic connection between the middle ear and the inner ear, increased pressure in the middle ear will be transmitted to the perilymphatic space in the inner ear, which produces an abnormal stimulus and causes vertigo and nystagmus.

Peripheral Retrocochlear Syndromes

Vestibular schwannoma: A second category of disease is a peripheral type of vertigo but with retrocochlear hearing loss (i.e., patients are found to have poor speech discrimination) Such patients should have an image of the inner ear, preferable an MRI. If an MRI cannot be obtained (e.g. pacemaker), a CT scan with thin cuts through the inner ear is also very useful.

It is important to recognize the presence of a tumor while it is still contained within the internal auditory meatus and thus easily surgically resectable or treated with some form of radiosurgery. Vestibular schwannomas (often incorrectly called acoustic neuromas) are histologically benign tumors, but they can become quite dangerous by position. If a vestibular schwannoma is allowed to grow into the brainstem, treatment requires a posterior fossa craniotomy, with significant morbidity and even some mortality. *Any patient with a history of progressive hearing loss should at some time during the evaluation have a careful auditory examination, and if any retrocochlear characteristics are found, a brain image with careful views of the internal auditory meatus should be obtained.*

Central Lesions

The last category of vertigo is central disease (i.e., patients with vestibular symptomatology that does not meet the criteria for peripheral disease). This group includes patients with vertical nystagmus, a vertical skew or bilateral nystagmus in the same position of the head. The horizontal head thrust maneuver is typically not abnormal in these patients.

Drugs: All drugs that act by intoxicating the reticular activating system in the core of the brainstem—including all anticonvulsants, all sedatives, and some sleeping pills—will by their nature produce nystagmus in two different directions in the same position of the head. When the patient looks to the right, the nystagmus beats to the right. When the patient looks to the left, it beats to the left. Overdosage can produce vertigo. Most sedatives (e.g. benzodiazepines) cause this type of nystagmus.

The fact that the lesion is central does not necessarily mean that it is serious. In fact, the appearance of this form of nystagmus may prove that a given drug (e.g. phenytoin) is in the therapeutic range. Such patients should be asked specifically about their use of drugs, including alcohol; before any invasive studies are performed, it is useful to order blood and urine toxic screening.

Demyelinating illness: Demyelinating illnesses, such as multiple sclerosis, can and often do produce vertigo, presumably because there are lesions somewhere in the vestibular system in the brainstem. Although such vertigo usually has characteristics that indicate a central lesion, occasionally it can resemble peripheral vertigo and be misdiagnosed as vestibular neuritis. If the same patient returns a year later with optic neuritis, it would be clear in retrospect that the first disorder was due to multiple sclerosis.

Vascular disease affecting the brainstem: In approaching vascular disease affecting the brainstem, it should be remembered that the most common manifestation of vertebrobasilar insufficiency is vertigo, but vertigo is almost never the *only* manifestation. Such patients can also be expected to complain of double vision, weakness of the limbs, sensory loss, dysarthria, and dysphagia. It might be possible for disease of the small branch of the vertebral artery to produce vertigo as its only symptom, but in such instances there is no specific or emergency therapy anyway. Patients with these syndromes have a negative horizontal head thrust maneuver, but may show bidirectional nystagmus in a single position of the head and/or a vertical skew deviation as judged using the alternate cover test.

Disorders of the temporal lobe: Temporal lobe seizures arising from trauma, tumors, or prior strokes can, as one of their manifestations, produce vertigo.

Migraine is strongly associated with vertigo. About 10% of patients with vertigo will ultimately be found to have migrainous vertigo. The reverse association is even more common. The majority of migraineurs have a history of motion sickness, which is physiological vertigo, and some patients have vertigo as the only aura of migraine. Episodes of vertigo lasting about 20 minutes, with or without associated headache, should raise this possibility. Most patients who have years of episodic vertigo without tinnitus or hearing loss probably have this syndrome. Whether migraine can actually damage the vestibular system is an open question. Often a therapeutic trial with prophylactic anti-migraine medication is required to make this diagnosis.

Treatment of Vertigo

Antiserotonin- and antihistamine-type

There are three categories of drugs for treating true vertigo. Anticholinergic and antihistamine-type drugs include dimenhydrinate, diphenhydramine, meclizine, and cyclizine. All of these drugs are effective if the dosage is adequate—about 50 mg every six hours. They produce major sedation as their side effect, but this is often of no concern. In patients in whom drowsiness is a serious problem, modafanil or methylphenidate may be used in concert with the anticholinergic drug. Vestibular sedatives work by suppressing the vestibular system thereby re-establishing balance between the two sides. Chronic use of these drugs is not advised as it will produce a vague sensation of dizziness, often described as imbalance, caused by loss of the normal proprioceptive function of the vestibular system.

DRUGS USEFUL IN SYMPTOMATIC TREATMENT OF VERTIGO

	Duration of Activity	Useful Adult Oral Dosage	Sedative Effects	Other Modes of Administration
Ethanolamines				
Dimenhydrinate	**4-6 hr**	**50 mg ev. 6 hr**		**Rectal IM, IV,**
Diphynhydromine	**4-6 hr**	**50 mg ev. 6 hr**		**IM, IV**
Piperazines				
Meclizine	**12-24 hr**	**25-50 mg ev. 6 hr**		
Cyclizine	**4-6 hr**	**50 mg ev. 6 hr**		**Rectal, IM**
Phenothiazine				
Promethazine	**4-6 hr**	**25 mg ev. 6 hr**		**Rectal, IM, IV**
Belladonna alkaloid				
Scopolamine	**4 hr**	**0-6 mg ev. 6 hr**		**SC, IV**

+ Mild ++ Moderate

The phenothiazines: promethazine

Promethazine is the only phenothiazine that works against the nausea associated with vestibular imbalance and vertigo. Other phenothiazines, useful for chemical nausea, are of no help whatsoever in this setting. Promethazine may be effective primarily because it is an anticholinergic, not because it is a phenothiazine It is useful also because it can be given together with the anticholinergic drugs and may be administered by a non-oral (e.g. rectal) route. A combination of promethazine and antihistamine is particularly effective for acute vertigo.

Beladonna alkaloids

A belladonna alkaloid, usually scopolamine, is used only for severe recurrent vertigo (e.g., in difficult cases of Ménière's disease) because it is a dangerous drug with many cardiovascular and psychiatric side effects. Transdermally absorbed scopolamine, although helpful for motion sickness, is of inadequate dosage for use in treating most acute vestibular syndromes. Many otoneurologists use benzodiazepines in addition to the anticholinergic drugs. These may have a specific anti-vertigo effect or may be acting on the almost universal associated anxiety in severely vertiginous patients.

Non-Vertiginous Types of Dizziness are treated depending on the specific diagnosis. In near syncope, the treatment may be as simple as discontinuing the precipitating drugs or may require a more specific therapy of the autonomic insufficiency (e.g. midodrine, an alpha adrenergic agonist) or a simple maneuver, such as leg crossing with thigh clenching, which simply squeezes blood out of the extremities to restore cerebral blood flow. In patients with disequilibrium, one would treat the underlying disorder, such as neuropathy, myelopathy or Parkinsonism. For anxiety, confident reassurance alone may be adequate with or without concomitant use of anxiolytic drugs.

Summary: To evaluate dizziness, one must first decide whether it can be categorized as near-syncope, disequilibrium, ill-defined light-headedness, or vertigo. If it is vertigo, vestibular and auditory testing will allow one to place the patient into one of three categories: peripheral cochlear disease, peripheral retrocochlear disease, or central disease. When this distinction is made, one can create a reasonable differential diagnosis and arrive at the likely diagnosis. Some of these disorders (e.g., vestibular schwannoma) require specific evaluation and treatment, whereas others have a benign natural history and require only symptomatic relief for the duration. Symptomatic therapy of vertigo is straightforward and makes use of the three categories of drugs discussed.

Selected Reading

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